CHAPTER 4

Mental Intensity and Your Heart

Jeffrey L. Boone

INTRODUCTION

This chapter discusses several concepts integral to the prevention of the early onset of heart disease. With present technolo advancement, early detection and primary prevention of cardiovascular disease are possible and even mandatory. The conc the "Four Horsemen" of mental intensity can provide a better understanding of key disease mechanisms or triggers for a hea attack. Traditional risk factors have been discussed earlier but added to these should be the concept of mental intensity. Thr the use of Mental Office Stress Testing, early detection of mental intensity can be provided and serve as a primary preventio initiative.

STRESS TRIGGERS FOR HEART PROBLEMS

Firearms lie dormant and seemingly harmless until the trigger sets the firing mechanism into action. However, despite a func firing mechanism, no one can get hurt as long as the chamber holds no cartridges. In a similar manner, mental intensity can a triggering effect on cardiovascular health. Hormones associated with mental intensity (i.e. noradrenaline) surge through th bloodstream of a senior executive during stressful times of the workday, acting as potentially lethal "triggers" for cardiovascu events. At age 35, this same executive could adapt to the response caused by noradrenaline because the chamber of the fir was empty (i.e. cardiovascular risk factors had not been around long enough to make the heart vulnerable). The stress trigge harmless because it fired in a heart that had not yet been affected by a lifetime of risk factors.

The cardiovascular risk factors of abnormal cholesterol, high blood pressure, diabetes, smoking, obesity, and limited aerobic physical activity act as bullets, waiting for the trigger (i.e. noradrenaline) to set off an acute cardiovascular event such as a h attack. Later in life, say, at age 55 or older, when the senior executive is confronted with similar tasks demanding high intens is now finally vulnerable to the "loaded" gun. In a sense, he is playing Russian roulette with the susceptible heart. Advanced strong family history of cardiovascular disease, and being of the male gender increase the heart's vulnerability to the long-te effects of noradrenaline. Logically then, we can conclude that blood pressure, cholesterol, and smoking do not directly kill; th simply establish an environment of increased vulnerability, arming the mechanisms that potentially can result in cardiovascu death.

Noradrenaline, along with other stress chemicals (e.g. cortisol), triggers cardiovascular events (i.e. heart attack or sudden ca death) through four separate mechanisms. These mechanisms, referred to as the "four horsemen" of mental intensity, are responsible for laying a foundation that results in adverse cardiovascular events. These mechanisms include:

- Plaque formation and rupture (i.e. coronary atherosclerotic plaque vulnerability)
- Blood clot formation (i.e. coronary thrombosis)
- Electrical abnormality (i.e. cardiac arrhythmia)
- Muscular abnormality (i.e. thickness, stiffness, or stretching)

THE FOUR HORSEMEN OF MENTAL INTENSITY

Plaque Formation. Coronary atherosclerotic plaque is the most notorious killer of our time. Stimulated by risk factors, the pla development occurs insidiously over many years, eventually corroding or clogging the arteries that supply blood to the heart major coronary arteries supply the heart muscle with the blood and oxygen necessary for appropriate function. The left main coronary artery (one) branches off the aorta and divides into the left anterior descending coronary artery (two) and the circur coronary artery (three). The right coronary artery (four) branches off the aorta on the opposite side to supply blood and oxyg the right side of the heart. Together, these four arteries provide nourishment to the tireless heart muscle.

Over many years, these arteries can become occluded (blocked, in lay terms) by atherosclerosis—a scar-like sludge of cells cholesterol, blood clot material, and inflammatory factors. The primary concern with atherosclerosis arises during the later st of development, when coronary blood vessels can become severely occluded. This occlusion eventually becomes severe er to restrict blood flow, causing coronary ischemia (i.e. reduced blood flow to the heart muscle) and angina pectoris (i.e. chest during exertion). Aggressive medical strategies, using medications and other procedures such as angioplasty or bypass surg are often pursued at this point to restore coronary blood flow. These important strategies can be life- saving since, in additio

controlling angina pectoris by restoring the flow of blood to the heart, they also reverse coronary ischemia. However, managing coronary atherosclerosis during the late stages by focusing primarily on the improvement of coronary artery blood flow does not address the majority of the morbidity (i.e. suffering) and mortality (i.e. death) associated with coronary artery disease.

In the 1990s, research in cardiovascular disease revealed that up to 70% of heart attacks occur in coronary blood vessels only minimally occluded with atherosclerotic plaque.[1] Therefore, it appears that only 30% of heart attacks occur in association with more severe blockages (i.e. greater than 50% occlusion of the vessel lumen or inner diameter). The coronary arteries that are severely occluded often result in angina and abnormal tests (i.e. on exercise test or electrocardiogram). This paradigm of evaluating atherosclerosis centers around blood flow limitation, while treatment is focused primarily on restoring blood flow.

Amazingly, however, more than two out of three heart attacks occur in association with coronary arteries that are not significantly blocked from a blood flow perspective. These are blockages that do not cause chest pain and do not make treadmill tests abnormal—yet they are very lethal! How can this be? It now appears that blood flow through the coronary artery is only a small part of the medical problem associated with coronary atherosclerosis. In fact, almost all heart attacks are found in association with a coronary plaque disruption (i.e. fracture or rupture of the vulnerable plaque) and coronary thrombosis (i.e. a blood clot adhering to the exposed vulnerable plaque). Therefore, plaque detection and stabilization define a new paradigm for the treatment of atherosclerosis. More and more, the traditional standard of care is being seen as "too little too late."

Coronary plaque is susceptible to disruption, rupture, or fracture especially in its early stages of development. The "young" coronary artery has a perfectly smooth internal lining conducive to rapid blood flow through the heart arteries with amazingly little shearing stress on the vessel wall. However, over the years plaque develops (often quickly in smokers and those with abnormal cholesterol, high blood pressure, diabetes, abnormal stress response, and poor physical condition), adversely affecting the smooth lining of the coronary artery. The plaque that develops on the innermost lining of the coronary arteries disrupts the integrity of the blood vessel, which then becomes vulnerable to unstable cracks and crevices. At this point, the circular muscles that surround the blood vessel wall lose the ability to relax sufficiently to handle the blood pressure surge in the coronary artery during times of mental and physical stress. Inflammatory cells (i.e. those that cause redness around a cut) weaken the plaque at its junction with the normal areas left in the vessel walls. Liquid cholesterol material lurks under the surface in pockets ready to burst. The progression of atherosclerosis is thus an evolving, percolating, scarring, and growing process. The developing plaque often is unstable and vulnerable to rupture and fracture if triggered by noradrenaline, which further endangers plaque stability during stress by accelerating hemodynamic (i.e. shearing) and vasoconstrictive (i.e. vessel constricting) forces on the coronary artery plaque.

These forces produce minimal effects on the plaque during sleep or times of low stress. However, when the afflicted person awakens and enters the workplace, his or her noradrenaline levels surge, endangering the susceptible plaque with the forces previously described. Not surprisingly, almost nine out of ten heart attacks are associated with plaque disruption. Over two-thirds of these occur in blood vessels that are only minimally blocked and that thus fail to generate warning chest pains. Even so, they are exquisitely vulnerable to the "trigger" of stress.

Blood Clot. In a deadly progression, the exposed surface of the fractured coronary plaque produces a coronary thrombosis or clot. For centuries, accelerated blood clotting during intense mental stress (e.g. combat) was a lifesaving part of the instinctive fight-or-flight response. Warriors wounded by sword, axe, arrow, or bullet better survived their wounds due to the aggressive clotting stimulated by stress chemicals (i.e. noradrenaline). For example, many of our ancestors survived attack by tomahawk and bear claw because of the clotting stimulated at the wound to prevent bleeding and promote healing. Our forefathers often survived due to the life-saving benefits of the surging stress chemicals accompanying the alarm response of conflict.

The senior executive today is rarely confronted with tomahawks or bear claws, but noradrenaline levels still surge as they did in our forefathers. The blood still clots quickly when you are loaded with the noradrenaline of workday accomplishment and challenge. Clotting components activate to make the clot larger and more tenaciously adherent during the intense stress of job strain—a good response if your job is wrestling grizzlies. However, today’s wound is not a sword to the flank, but rather a ruptured plaque in a coronary artery. Blood responds today as it did centuries ago with aggressive clotting at the site of plaque rupture and the tenacious clot protecting the wound in the coronary artery wall. Unfortunately, the coronary clot may also occlude the entire blood vessel, thereby blocking blood flow to areas of the heart. If the clot is not eliminated, much of the heart muscle downstream will die as the heart attack spreads. How ironic that the response to mental intensity designed to keep us alive during times of stress is today considered a major health threat in our society!

The clots have moved from the status of protectors to killers during the 20th century. In earlier times, life was filled with physical adventure pulsating with appropriate surges of stress chemicals such as noradrenaline. Today’s senior executive is not "tomahawked" or "bear-clawed" in a physical sense. Accelerated thrombotic (i.e. clotting) activity is therefore not

needed during the cerebral white-collar stress of the modern era. As a result, the clot becomes a killer, not a "savior." Nearly 90% of all heart attacks occur with coronary plaque fracture (the "new" wound) and coronary thrombosis (the "internal" clot) blocking blood flow to the heart muscle.[2] These processes result from the deadly intersection between the vulnerable plaque disruption, caused by the shear forces and vessel constriction promoted by the noradrenaline of work stress, coupled with the clotting forces of stress working to seal the wound. Unfortunately, instead of promoting healing, the clot clogs the coronary artery. And, to repeat, at least two of three heart attacks that occur in this manner are found in coronary arteries that were only minimally occluded before the plaque fracture (i.e. the kind of lesions that would not be detected with a treadmill test). [3]

Electrical Abnormality. The beating of the heart is controlled by cardiac tissue, which is specially adapted to convert chemical signals into electrical impulses that cause the heart muscle to contract in a coordinated fashion. The rhythm of the heart is very sensitive to noradrenaline. In the healthy heart, the pulse quickens during times of physical or mental stress and slows with rest. Usually, if the heart is healthy, the pulse is regular and rhythmic, whether fast or slow. As the heart ages or becomes vulnerable through cardiovascular risk factors, it becomes more susceptible to electrical stimulation by the noradrenaline of stress. A few premature beats generated in the heart atria (i.e. blood receiving chambers) or the ventricles (i.e. blood pumping chambers) each day are often considered normal in an otherwise healthy heart. However, 24-hour EKG monitoring studies show that these premature beats occur in a healthy heart most frequently during the waking hours when noradrenaline levels are highest compared with other times of the day.[4] Similarly, noradrenaline triggers advanced electrical disturbances (arrhythmias), which can be severely compromising to the heart—especially if the heart is also affected by coronary plaque or cardiac muscular thickness, stiffness, and stretching.

Heart Muscle Abnormality. The chambers of the heart work best when they are of normal size. When the chambers enlarge (e.g. left ventricular enlargement and left atrial enlargement) or become thick (e.g. left ventricular hypertrophy) and stiff (e.g. left ventricular diastolic dysfunction), the ability of the heart to pump blood efficiently can be adversely affected. Interestingly, the process of muscular change within the heart arises from many variables, not the least of which is stress-produced noradrenaline. Certainly, heart muscle can thicken in response to pumping against high blood pressure. However, many patients with high blood pressure have no deleterious cardiac muscle changes. In fact, numerous patients who have never had high blood pressure have been found to have dangerous thickening of the heart.[5] Recent data suggest that blood pressure measured during mental stress provides good predictions of abnormal cardiac muscle changes (i.e. thickness, stiffness, and stretching). Blood pressure measured during mental stress also provides the basis for deductions concerning the amount of noradrenaline produced by the subject in generating those blood pressures. Both blood pressure and noradrenaline thicken, stiffen, and stretch the heart over time. Once this occurs, the heart is more susceptible to daily stress. This situation will further complicate any existing coronary atherosclerosis and cardiac arrhythmia conditions.

CIRCADIAN RHYTHM OF CARDIOVASCULAR EVENTS

Each day throughout America, a convenient cycle of noradrenaline production occurs in the population, which helps us understand the impact of stress on the heart. Noradrenaline levels plummet as we sleep and surge as we awaken and enter the workplace. Therefore, the four horsemen of mental intensity should be quiet at night and galloping during the workday (which most Americans experience during the daylight hours). The incidence of heart attacks, stroke, and sudden cardiac death shows that a reproducible pattern occurs each day, with greater numbers of events occurring during the workday and fewer occurring at night. This type of pattern is known as circadian (circa = about; dies = day) rhythm because it happens regularly over a 24-hour period.

In the susceptible patient, the gun is always loaded but the trigger is activated only when noradrenaline "squeezes" the plaque, clot, electricity, and/or heart muscle thus producing the cardiovascular event as its deadly discharge. Countries with the siesta experience a lull in cardiovascular events during those two hours of rest, which serve to relax the trigger finger. In nightshift workers, the noradrenaline pattern is reversed from that of most of the working population. The death rate is accelerated in these workers at night, when the noradrenaline trigger activates upon awakening.

These instances of increased death occur because of the noradrenaline activation of coronary plaque fracture, coronary thrombosis, cardiac arrhythmia, and/or left ventricular diastolic stiffening in the susceptible heart. The famed Framingham Heart Study, which has monitored several generations of people to determine risk factors and trends in cardiovascular disease, has confirmed this circadian rhythm of cardiovascular events.[6]

INTENSITY VS. STRESS IN THE WORKPLACE

The health implications of stress are very difficult to ascertain with regard to any specific group, business, or individual. Stress generally occurs when the values, beliefs, and goals of the individual do not mesh with the values, beliefs, and goals of the organization. However, preoccupation with such dissonance-induced stress obscures the most critical and ubiquitous

effects of stress at work—the cardiovascular and physiological consequences. Therefore, the term "intensity", which implies the interaction of hard work and noradrenaline, is often preferred over "stress." For the most part, intensity has an upbeat, positive connotation. When applied to the individual or the corporation, the term intensity seems complimentary, invoking thoughts of pride and accomplishment. At its core, intensity is a good characteristic for the individual worker and for the company. Medical, physiological, and cardiovascular concerns regarding occupational intensity develop only when the demands and pace of job and life accelerate to the point of deleterious physiological effect, or when the age-health-risk profile of the individual worker worsens to the point that previously well tolerated tasks are now of medical concern.

To center the discussion on "stress" evokes negative connotations, often seeming to imply weakness, failure, and burnout. Such perceptions unfortunately permeate corporate culture and may actually interfere with successful stress management from a psychological perspective, whereas the subtle shift toward discussion of intensity issues and susceptibilities takes on positive tones. The focus on intensity can thus be a better starting point, dealing more with heart attack prevention and related medical initiatives than with nervous breakdown issues. Once the program is established with intensity and heart attack prevention as its anchors, more subtle psychological issues can be woven in.

CORPORATE INTENSITY VS. INDIVIDUAL INTENSITY

An individual's own unique approach to life's stresses as well as the person's age, sex, habits, and susceptibilities determine the cardiovascular consequences of mental intensity. Yet, an individual's environment overlaps the broader universe of family, neighborhood, workplace, and government that further influences cardiovascular health as related to mental intensity. Thus, both individual and corporate responses need to be established to combat the cardiovascular consequences of mental intensity. Fortunately, the same intensity-control strategies that assure the healthy individual also seem to beget the healthy corporation.

Descriptions of the effects of noradrenaline on larger populations are rife in the literature:

- New Yorkers' death rates from heart attack are 55% higher than the average rates for the United States as a whole.[7] The city's electric aura stimulates an outpouring of noradrenaline which triggers a much higher rate of adverse cardiovascular episodes than San Diego, for example. When New Yorkers travel outside the city they die of coronaries at a rate 20% lower than if they had stayed at home. Interestingly, the pace of life in the Big Apple even affects tourists. Their heart attack death rate in New York is 34% higher than the U.S. average for visitors dying outside their home cities. Typically, visitors to other cities among America's ten largest have only about a 1% higher rate of cardiovascular death. The environment of New York is filled with pressure that drives the noradrenaline trigger, causing potentially adverse stimulation of cardiovascular plaque, coronary thrombosis, cardiac arrhythmia, and cardiac muscle. Thus the same heart moving from San Diego to New York finds a new environment of exciting but dangerous noradrenaline and other stress chemicals! Most people love New York just as most executives love their jobs and their corporations. However, healthy awareness of the cardiovascular consequences of exciting, upbeat, and positive intensity is necessary to safely pursue the opportunities offered in life.

- Aerospace workers in the 1960s had an alarming rate of sudden death. In fact, foul play was suspected for a time as the space race with the Soviets heated up. Eventually, it was discovered that the aerospace engineers were literally killing themselves in trying to meet the intense, urgent, and complicated demands of space exploration. [8]

- In Japan, "karoshi" appears on death certificates—meaning "working oneself to death."
- Heart attacks accelerated during the Northridge earthquake and aftermath.
- Sudden death increased as Tel Aviv awaited the missile attacks from Iraq during the Persian Gulf War.
- Most cardiovascular death occurs at work, especially before noon, rather than athome at night, thus illustrating the relatively greater effects on the noradrenaline trigger in a workplace setting.[9]

Recognizing and anticipating mental intensity at the corporate level should stimulate appropriate protective responses. The negative consequences of work site intensity can be proactively mitigated with an emphasis on protective exercise, nutrition, weight control, and stress hygiene/management, coupled with selected screening and prevention strategies.

SCREENING ASSESSMENT––MENTAL OFFICE STRESS TESTING

It is natural for people to arise and tackle the challenges that each day has to offer. The noradrenaline surge prepares us for this task by readying our bodies and minds for action. This is a great part of life and work; yet, it is important to detect when the intensity of work becomes dangerous and worthy of protective attention.

Blood pressure can be a simple, effective marker for stress and intensity susceptibility and sensitivity—but not the usual blood pressure reading you receive in the doctor's office. True, this is an important reading that defines the disease hypertension (blood pressure readings above 140/90 mm Hg). High blood pressure, even at rest, is an indication that your body has more noradrenaline than it can tolerate.

But the particular blood pressure readings needed for intensity measurement are "stressed-induced." It is important to perform them on each individual in the corporation. Blood pressures measured during mental stress and intensity are better predictors of the potential harm that can be caused by noradrenaline. Stress-related blood pressure surges predict coronary vessel constriction and shear forces acting on undetected coronary plaques. The magnitude of the blood pressure surge during stress also predicts accelerated blood clotting through noradrenaline-related effects on platelets and other clotting mechanisms. Cardiac electrical instability and cardiac muscular changes are predicted by stress-induced blood pressure surges as well. Blood pressures taken during stress, therefore, begin to identify those susceptible to the four horsemen of mental intensity.

Devices called Ambulatory Blood Pressure Monitors (ABPM) are now available to monitor blood pressure throughout the day. Measurements are taken multiple times every hour and the patient records his or her daily activities in a diary. With this information, the blood pressure response can be correlated with particular stressful activities. However, ABPM is impractical for widespread medical and corporate application because of the large number of persons who require screening. Instead, another simple mental stress testing method can be used for the frequent and widespread screening of persons to establish their susceptibility to the cardiovascular consequences of mental intensity. This testing method is like a mental treadmill that measures the impact of life intensity on the cardiovascular system. I call it the Mental Office Stress Testing Protocol, or M.O.S.T. Protocol. Stressing patients in an office setting produces blood pressure and heart rate responses approximating those experienced by individuals during the more intense times of the workday. The M.O.S.T. Protocol can be performed in less than 5 minutes and is inexpensive in terms of equipment and space. In fact, it could even be performed right at the work station.

The M.O.S.T. Protocol should be used routinely during medical or health screening visits. However, it should be administered more frequently for all individuals who

- are over 40 and male (however, women with blood pressure reactions to intense stress also exhibit increased atherosclerosis[10]),
- have a history of high blood pressure,
- have elevated cholesterol levels,
- have diabetes,
- are smokers,
- are obese,
- live a sedentary lifestyle,
- have a family history of heart disease, stroke, high blood pressure, abnormal cholesterol, and/or diabetes,
- work in high-intensity occupations and /or have specialized risks (i.e. lipoprotein lipase activity, small dense LDL, homocysteine, chlamydia titers, uric acid, etc.)

The M.O.S.T. Protocol, which simulates daily stress and correlates well with ABPM measurements, is performed as follows:[11]

Step 1. Baseline Assessment: Measure sitting blood pressure and heart rate.

Step 2. Relaxation Assessment: Ask patient to relax and breathe deeply and consistently for 5 breaths over 15 seconds. Take blood pressure and heart rate measurements at the conclusion of the final deep breath.

Step 3. Stress Assessment:

- Mathematics challenge—Have patient begin successive verbalized subtraction of 7, starting with 777 (i.e. 777, 770, 763, 756, etc.). Take blood pressure and heart rate measurements after 1 and 3 minutes of subtraction.

- Cold pressor challenge—Insert patient's hand past the wrist in ice water. Take blood pressure and heart rate measurements in opposite arm within 60 seconds.

- Grip challenge—Have patient hold 30% of maximum handgrip. Take blood pressure and heart rate measurements in opposite arm within 60 seconds.

M.O.S.T. Protocol Parameters include any blood pressure reading above 160 systolic or 95 diastolic and/or heart rates above 75. Exceeding any of these parameters may indicate increased cardiovascular reactivity to mental stress and designate a potential hot reactor.

Screening with the M.O.S.T. Protocol advances our ability to identify people at risk for intensity-related cardiovascular problems. On the basis of measurements gleaned from the protocol, evaluation strategies for determining the degree of risk can be developed such as those described below, along with health maintenance programs carefully tailored to the condition.

EVALUATION OF THE MEDICAL CONSEQUENCES OF MENTAL INTENSITY

An abnormal M.O.S.T. Protocol finding, especially when coupled with any other risk factors, requires further medical assessment. An elevated blood pressure surge during brief periods of mental intensity may indicate a problem in one or more of the potentially lethal four horsemen of cardiac concern—plaque, clot, electricity, and muscle.

Optimal noninvasive evaluation strategies can be implemented in patients at highest risk while others determined to have less immediate risk (i.e. those who are younger with fewer compounding problems) can bypass more costly testing and move directly to preventive treatment strategies along with lifestyle modification, adding medication strategies if needed. The noninvasive evaluation strategies are painless, relatively inexpensive, and have an immediate impact in ascertaining how best to manage the cardiovascular consequences of mental intensity. The strategies for each of the four horsemen are as follows:

Plaque. An abnormal M.O.S.T. Protocol indicates that the linings of the coronary arteries are being bombarded by noradrenaline during times of intensity. These arteries are then being subjected to vascular narrowing and accelerated shear forces. Simultaneously, factors that promote blood clotting are being stimulated. Remember that two-thirds of all heart attacks occur when non-flow-limiting coronary plaques fracture or rupture. These fractures are then covered with stress-stimulated clotting material, thus occluding the coronary artery.

But who has coronary plaque? If we knew, we could specifically treat the plaque- afflicted individual who also shows M.O.S.T. Protocol abnormalities. In such patients, aggressive treatment should be given to stabilize the plaque. Identifying patients who have non-flow-limiting plaques can be difficult and expensive. Treadmill tests assess the possibility of flow-limiting coronary plaque. However, all coronary plaques can be dangerous, especially for people with M.O.S.T. Protocol abnormalities. Fortunately, a new plaque-detecting device has become available. Called Electron Beam (Ultra Fast) Computerized Tomography (EBCT), this simple, safe, painless test takes only a few minutes to search the coronary arteries for the presence of plaque. To be more precise, the EBCT actually detects the calcium material present in coronary plaque structure. It provides the most accurate method currently available to determine whether an individual has asymptomatic atherosclerosis (see Chapter 3).

Once plaque is detected by EBCT, a treadmill test can determine whether the plaque limits blood flow and point to cardiologic interventions that need to be considered. Most often at this stage, however, the coronary plaque is not limiting coronary blood flow. But since plaque can kill all the same, aggressive noradrenaline and plaque stabilization efforts need to be incorporated into long-term medical management, including the treatment strategies described below. Remember, a normal treadmill test (and related tests) confirms the absence of ischemia (i.e. coronary blood flow limitation) but not the absence of plaque. Abnormal M.O.S.T. Protocol findings warn for potential plaque danger. The EBCT detects calcified plaque. When detected, plaque formation should be halted and reversed.

Clot. When you wake up and go to work, your blood clotting quickens and becomes more tenacious. This is not good in routine circumstances, and it is especially dangerous in the presence of plaque. Further concern with clotting arises if a poor response is demonstrated with the M.O.S.T. Protocol. Blood pressure spikes during times of mental intensity signal that clotting may be more aggressive than usual. The abnormal plaque detected on the EBCT heightens the need for clot control because a plaque fracture could occur in the coronaries, providing a susceptible site upon which an occluding coronary thrombosis (clot) could develop. However, further clot testing is not usually necessary. Your physician may merely prescribe an aspirin per day to bring the clotting back to normal. If you show no plaque on the EBCT, a high dose of Vitamin E (i.e. 400 to 800 IU) and various healthy lifestyle choices should alleviate your clotting concern.

Electricity. Noradrenaline from the intensity of life can trigger all forms of arrhythmias. Therefore, rhythm disturbances are much more likely to occur during the stress of the workday. A heart with abnormal muscular thickness, stiffness, and/or stretching, as well as one with excessive atherosclerotic plaque development, is especially susceptible to arrhythmia.

Palpitations, which are symptomatic signals of arrhythmia, also require further medical evaluation. An electrocardiogram (EKG) measures the electrical activity of the heart and sheds light on cardiac electrical stability. The EKG readings are standardized with certain rates, waves, spaces, and intervals determined as normal while at rest as well as while exercising. Deviation from a normal rhythm requires further medical evaluation. Twenty-four-hour monitoring of cardiac electrical activity by a special type of EKG can be very useful in evaluating the effects of noradrenaline and mental intensity on palpitations and arrhythmia, especially when coupled with resting and exercise EKG measurements.

Even the most basic assessment of heart rate can indicate the need for action. For example, according to the Framingham Heart Study, a fast pulse (i.e. 80 beats or more per minute at rest) in a worker with high blood pressure is a signal of excessive sensitivity to noradrenaline and is a marker for premature adverse outcomes such as heart attack.[12] Simple preventive strategies can be adopted at this early time to forestall problems.

In later life, cardiac electrical problems arise as a result of the weakening in coronary circulation or the cardiac muscle, thus aggravating these vascular and muscular concerns. Arrhythmias arising in a heart with totally normal cardiac muscle and unoccluded coronary circulation are rare and often congenital. However, they too can be triggered by noradrenaline. The M.O.S.T. Protocol helps predict these and other arrhythmic sensitivities.

Muscle . The heart muscle is designed to work tirelessly to transport blood throughout the body in a rhythmic series of contractions that move this life-sustaining fluid through four cardiac chambers to parts distant. The upper chambers are primarily holding tanks (the atria) which receive blood from the body. These upper chambers then send their contents through one-way valves into the pumping chambers (the ventricles) below. The transit valves close and the exit valves open while a forceful contraction of the cardiac muscle ejects most of the contents from the ventricles. The left side of the heart is the most powerful, since the left ventricle, which sends oxygenated blood out to the body, is the primary muscular pumping force of the heart. Blood vessels (the coronary arteries) on the surface of these chambers branch to supply fuel to the working heart muscle. Noradrenaline stimulates these blood vessels to become clogged with atherosclerotic plaque and occluded through coronary thrombosis.

Noradrenaline also helps cause the cardiac muscle itself to become stretched (left atrial enlargement, left ventricular enlargement), stiffened (left ventricular diastolic dysfunction), or thickened (left ventricular hypertrophy or LVH) after years of intense stress. In fact, a heart can be dangerously thickened even with no plaque buildup in the arteries. The M.O.S.T. Protocol provides excellent screening assistance in identifying potential for cardiac muscular abnormality. Blood pressure measured during mental stress challenges is the best clinical predictor of LVH.[13] The M.O.S.T. Protocol blood pressure readings identify noradrenaline-related blood pressure surges, which thicken the heart.

Testing for cardiac muscular abnormality is very important. Echocardiography (cardiac ultrasound) should be considered whenever an adverse M.O.S.T. Protocol finding occurs. However, the most dangerous LVH is usually preceded by several years of cardiac muscular stiffness or diastolic dysfunction. This can be detected with a stethoscope, which picks up noise called an S4 gallop when the heart has stiffened due to too much noradrenaline exposure over the years. An adverse M.O.S.T. Protocol finding plus an S4 gallop indicates the need for echocardiography. Echocardiography can be performed during a repeat performance of the M.O.S.T. Protocol to identify cardiac stiffness that might only occur during the actual stressful event. In that case, therapeutic intervention in the process could begin even earlier if indicated. We should note that a thick heart with LVH can occasionally be normal if the thickness is due to a good exercise program. A thick heart is considered normal if it is flexible and supple, thus lacking the stiffness associated with adverse responses to noradrenaline and blood pressure load.

In sum, the M.O.S.T. Protocol detects physiological stress susceptibility. Intensity intervention through many avenues then begins to heal the employee and, in the aggregate, the company as well. Treatment, as well as the evaluation and advanced testing previously recommended is multidimensional, moving from the simple to the complex: the earlier the intervention, the simpler the approach. The necessary degree of sophistication of the treatment and the testing is best determined when the additional screening factors of cholesterol, blood pressure, diabetes, body weight, genetic risk, and physical fitness are factored into the equation. An abnormal M.O.S.T. Protocol alerts the health care team to institute interventions at an even earlier point than other standard risk assessments.

LIFESTYLE INTERVENTIONS

Exercise. Aerobic exercise, the kind that makes your heart race and your breathing faster, is an excellent antidote for all the cardiovascular consequences of mental intensity. It benefits everyone but is especially important for those who fail on the M.O.S.T. Protocol, indicating they are especially sensitive to the noradrenaline of stress. Regular aerobic exercise is able to reduce the excessive noradrenaline of stress. Aerobic activity also beneficially affects plaque shear forces, heart rate and other arrhythmias, cholesterol profiles, excessive clotting, and heart musculature. Essentially, it is one of the few things that you can do to improve all of the cardiovascular consequences of mental intensity.

Many books have been written about proper exercise and its benefits. The best of these is the series by Kenneth H. Cooper, M.D., founder of the Cooper Aerobics Center in Dallas, Texas. His writings will be an excellent guide to both the individual and the organization for establishing exercise programming to develop "intensity heartiness."

Nutrition. Proper nutritional habits are an effective intervention for cardiovascular disease. A failure on the M.O.S.T. Protocol warrants additional nutritional efforts. Alcohol reduction, caffeine control, increases in dietary fiber (e.g. fruits, vegetables), reduced dietary fat, controlled calories, and avoidance of refined sugars can all assist in the battle. A diet high in potassium, magnesium, calcium, fish, and fiber, but low in calories, caffeine, alcohol, fat, sugar, and saturated and polyunsaturated fats (particularly hydrogenated) can combat the cardiovascular consequences of mental intensity.

In the past, advanced nutritional therapies have been the subject of ridicule from the medical profession. However, several important "nutriceutical" therapies have emerged with substantial medical support. Antioxidants such as Vitamin E and Vitamin C have been found to have a very favorable effect on reducing the oxidation of cholesterol within the coronary plaque. Vitamin E may also attenuate clotting. Folic acid reverses the potential coronary risk of elevated homocysteine. Vitamins B6 and B12 may be very useful during times of stress. As before, the Cooper Aerobics Center is a good place to start (along with Chapter 8 in this volume) for sound advice regarding cardiovascular and general nutrition as well as advanced nutritional therapies. Georgia Kostas, M.S., R.D., and her staff assist Dr. Cooper in the promulgation of appropriate nutritional advice for the individual and the organization.

Weight Control. Excess body weight and fat worsen M.O.S.T. Protocol scores. The problem should be addressed at both the individual and the organizational levels. However, weight and fat hardly need to be treated as separate problems if the nutrition and exercise programming are optimized.

Stress Management. Exercise, good nutrition, and weight control provide the basic physiological strategies for stress control programming at the individual and organizational levels. These efforts allow the stress to occur in a lean, sleek, well-oiled body that can stand a lot of punishment. Certainly, they also assist in improving the psychology of stress and intensity. However, the psychological aspects have not really been addressed until more formal stress management training has been instituted. It has been demonstrated that even simple sessions of abdominal breathing training can substantially impact an abnormal M.O.S.T. Protocol.[14] Additional efforts with progressive muscle relaxation, biofeedback, prayer, meditation, cognitive restructuring, and other more advanced techniques can provide further assistance where needed (also see Chapter 5 of this text). The writings of Dr. Cooper, Robert S. Eliot, M.D., Herbert Benson, M.D., Stephen Fahrion, Ph.D., and Redford Williams, M.D., provide excellent frameworks for the development of the psychological and stress management components of a "stress heartiness" program.

MEDICAL INTERVENTIONS

One of the great advances in preventive cardiology is the advent of multiple medications that can effectively forestall the progression of the cardiovascular consequences of mental intensity with few side effects. In the past, medications were viewed with suspicion if not disdain by many in the preventive arena. However, they have now become the cornerstones of heart attack prevention strategies, complementing and closely allied with lifestyle interventions.

Medicinal therapy should be used on all patients with any form of cardiovascular disease (i.e. even to include early M.O.S.T. Protocol failures). Obviously, the non-drug therapies described earlier should also be implemented. But if the intensity is high or the body susceptible, drug strategies should be implemented. Beta-blockers, commonly known as a type of medicine to treat hypertension, have now become the standard of care for advanced cardiovascular disease. Most cardiologists use beta-blockers to treat advanced plaque and electrical and muscular abnormalities of the heart. Why? Because beta-blockers "block" the effect of noradrenaline on the heart, thus protecting the very sick heart afflicted with advanced atherosclerosis, accelerated arrhythmia, and cardiac muscular failure.

Are beta-blockers the best choice? Certainly they are an excellent choice for patients who have just suffered a heart attack. However, for patients with high blood pressure, the preferred agent would control noradrenaline levels similar to beta-blockers, but without the potential bad side effects common with these agents. The beta-blocker, for example, may cause fatigue, depression, and impotence, and it is also problematic when the patient also suffers from high cholesterol and asthma.

Verapamil, a calcium channel blocker, is the drug of choice for many practitioners who treat high blood pressure. It lowers noradrenaline by 30% without the side effects of a beta-blocker.[15] Verapamil decreases clotting, lowers the heart rate, controls arrhythmia, reduces the shear forces on the coronary plaque, dilates coronary arteries, reverses cardiac muscular thickness, and effectively lowers resting blood pressure and controls M.O.S.T. Protocol blood pressures as well.[16] In fact, it is the only single drug that controls all four horsemen of mental intensity. Your doctor should consider verapamil as the cornerstone therapy for high blood pressure just as the cardiologist builds therapy for sicker patients around the beta-

blocker. Recently, verapamil has also been packaged in a stress-correlated delivery system in which the drug is taken at night with a delay coat around the pill that dissolves after 5 hours to release the majority of the drug into your system during the workday when it is needed the most.

Medicines called ACE (angiotensin-converting enzymes) inhibitors, as well as angiotensin receptor blockers (ARBs), also are outstanding for high blood pressure, especially when combined with verapamil. Alpha-blockers, diltiazem, low-dose diuretics, and other medications may be useful as well. Obviously, these are treatments to be discussed with your doctor but you will especially want to discuss the advisability of a noradrenaline strategy.

Interestingly, one of the most widely prescribed groups of medicine for high blood pressure is the dihydropyridine calcium channel blockers (i.e. amlodipine, felodipine, nifedipine, isradipine, nisoldipine, nicardipine, etc.). These drugs are highly effective at lowering blood pressure, but they actually increase noradrenaline levels by 17% according to a recent review of the medical literature.[17] This may explain why these drugs look good in medical studies only when they are used like a cardiologist uses them—always in combination with a beta-blocker to block the effect of increased noradrenaline levels. Verapamil plus an ACE inhibitor or ARB is a much better strategy for "trigger" control. The world's medical literature validates such choices where the goal of treatment is to save the heart, brain, and kidneys and not just lower blood pressure.

Significant advances in the management of elevated cholesterol have been made as well with the advent of drugs known as "statins." These drugs dramatically improve the cholesterol profile, but even more important they are powerful plaque stabilizers. Statins shrink coronary atherosclerotic plaques through the process of reducing the pooled liquid cholesterol swelling the plaque. This then reduces the inflammatory weakening of the plaque at its edges. The "hide" over the plaque is toughened and made more resistant to the shear forces and vessel constrictions that occur during the M.O.S.T. Protocol as well as during the intensity of the workday. Statin drugs are mandatory, I believe, if the M.O.S.T. Protocol is abnormal and there is plaque detected on the EBCT. Keep in mind that our goal is more urgently plaque stabilization than cholesterol lowering. Recent studies have shown that even people with normal cholesterol levels have fewer heart attacks when treated with statins. [18]

How about aspirin? This wonderful clot controller should be considered for administering after all M.O.S.T. Protocol failures. Remember, the same noradrenaline that raised the blood pressure also accelerated the clotting of blood. Taking aspirin must be discussed with a physician. The data are compelling, however, in terms of aspirin's benefit after both EBCT and M.O.S.T. Protocol failures. A recent study from the Harvard Hospital System confirmed that angry men had a higher heart attack rate than non-angry men. However, a sub-group of angry men taking aspirin regularly had heart attacks at a rate closer to that of non-angry men. The anger may have surged blood pressure and fractured plaque, but with patients taking aspirin, a coronary thrombosis or clot was less likely to form. [19]

Obviously, medication strategies can be critical to healthy intensity management. Thus individuals need to receive medication choices for noradrenaline and intensity management. However, I believe that involvement in medicine choices can be practical and important even at the organizational level. Medical education, physician discussions, and intensity-related medical goal-setting can and should be facilitated through organizational medical plans.

Medical management of the cardiovascular consequences of mental intensity is like the safety net under the trapeze act in a circus. The executive is on the trapeze facing challenges and new performance routines daily. The M.O.S.T. Protocol and other strategies indicate the executive's grip on the swing maybe slipping. Exercise physiologists, nutritionists, psychologists, and spiritual leaders are the trapeze instructors teaching life management skills. But when all else fails, the physician with his formulary of medications acts like the circus maintenance man strengthening the safety net to protect the executive while he or she learns how to better manage the trapeze.

SUMMARY CASE STUDY

The following case provides a real world example of the benefits of the approach presented in this paper.

John was an apparently healthy 54-year-old corporate executive trained in law. His degree served him well as he climbed the corporate ladder to secure a senior vice-presidency. He had no significant complaints of either mental stress or physical symptoms of cardiovascular disease. His health profile was normal on regular screening examinations each year. Treadmill tests were normal and his last cholesterol level was a low 169 mg/dl with 49 mg/dl of that being the protective HDL type of cholesterol. He did have high blood pressure, however, and his internist placed him on the calcium channel blocker amlodipine, reducing his resting blood pressure to a normal 135/85 mm Hg.

But this year his corporation recommended him for a much more aggressive preventive cardiology assessment centered on

intensity medicine. John participated in a new test called the M.O.S.T. Protocol, during which he was asked to subtract numbers in reverse order to create some stress. Amazingly, his blood pressure rose to 186/107 during this simple mathematics challenge, despite amlodipine therapy. He was told that the amlodipine was not the best drug to control factors related to the intensity of work.[20] The drug amlodipine (though effective at controlling resting blood pressure) was not a plaque stabilizer, clot stabilizer, electrical stabilizer, or intensity stabilizer, which control the "triggers" for cardiovascular events.

This information became especially important to John when another new test called the EBCT scan showed that he had four previously unknown places in his coronary arteries where atherosclerotic plaque was accumulating. Therefore, a switch from amlodipine to verapamil was recommended to provide better intensity control and plaque stabilization. Verapamil would reduce the shear forces on the plaques, as well as the powerful contractions of the heart during stress. It would also lower clotting and heart rate, unlike amlodipine.

The treadmill continued to be normal, so that there would likely be no need for invasive testing and/or intervention. However, since up to 70% of heart attacks occur in blood vessels that are not clogged enough to cause chest pain or show up as abnormal on a treadmill, plaque stabilization beginning with verapamil would be pursued even more aggressively with the addition of a statin drug to treat cholesterol. John asked why treat his cholesterol when it was only 169 mg/dl. He was told that the most important aspect of the statin drug was its action as a plaque stabilizer and plaque shrinker. Aspirin was added to cut down the possibility of coronary thrombosis. Advanced nutritional supplementation with Vitamins C, E, B6 and B12 plus folic acid were added to his daily regiment to combat oxidation, stress, and homocysteine (other aggravators of plaque).

An echocardiogram or ultrasound of the heart revealed thickened and stiffened heart walls, so an ACE inhibitor was added to the verapamil to further accelerate the return of the heart muscle to normal. This muscular improvement should occur in 6 to 12 months, eliminating the minor premature contractions noted on electrocardiographic monitoring during the treadmill.

A refresher course on proper nutrition and exercise, along with some executive hints about noradrenaline control in the workplace through relaxation breathing, completed the process. Repeat testing at intervals would document reversal or stabilization of the triggers—the four horsemen of intensity—of the cardiovascular consequences of mental intensity.

Sure enough, a year later, John was lean and fit. However, even more importantly, after the elapse of still another year, a repeat EBCT scan showed coronary plaque stabilization, a repeat echocardiogram demonstrated reversal of heart muscle thickness and stiffness, and a repeat treadmill EKG revealed the absence of abnormal cardiac electrical patterns. The four horsemen had finally dismounted.

ENDNOTES

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